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Evaluation of clinical and laboratory findings in adolescents diagnosed with polycystic ovary syndrome

Polycystic ovaries in adolescence

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Abstract

Aim: Polycystic ovary syndrome (PCOS) is a heterogeneous condition characterized by menstrual irregularities, hyperandrogenism, obesity, hirsutism, and psychological and psychosexual morbidity. This study aims to investigate the relationship between clinical, anthropometric, laboratory and ultrasonography findings in adolescents with the diagnosis of PCOS.

Material and Methods: Forty-one adolescent girls who were admitted to the Pediatric Endocrinology Clinic of Adana City Training and Research Hospital between January 2020 and December 2021 and diagnosed with PCOS according to the Rotterdam criteria were included in this retrospective cross-sectional study. Demographic data, hormonal and biochemical analysis of the patients were obtained from the files.

Results: The mean age of admission of the patients was 15.62±1.43 years, and the mean age at first menarche was 11.98±1.32 years. Oligomenorrhea (90.2%) was the most common admission complaint. Comparing the groups with PCOS with and without hirsutism, a significant difference was found in inhibin-B level (66±45 ng/dl vs 107±37 ng/dl; p=0.038). When comparing the groups with and without ultrasound findings compatible with PCOS, a significant difference was found in AMH level. (11.0±7.3 mcg/L vs 5.9±2.8 mcg/L; p=0.032). When overweight or obese patients with PCOS were compared with the normal weight group, the difference was significant in HOMA-IR (5.0±6.2 vs 1.5±0.65; p=0.009) and first menstrual age (11.3±1.3 years vs 12.7±0.75 years; p=0.008).

Discussion: In this study, while low inhibin-B was more common in patients with PCOS with hirsutism, AMH was found to be significantly higher in the group whose ultrasound findings were compatible with PCOS. These parameters can be investigated in the diagnosis of adolescent PCOS.

Keywords

Adolescent, Anti-Müllerian Hormone, Inhibin A, Inhibin B, Polycystic Ovary Syndrome

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Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrinological disorder of women of reproductive age, affecting 7-13% of them [1]. For adult women, diagnosis should be based on clinical, laboratory and ultrasonographic findings. From the clinical perspective, oligomenorrhea, androgen excess, hirsutism, and obesity are the most common findings. During the adolescence period, the diagnosis of PCOS is challenging because normal pubertal physiologic changes overlap with the PCOS diagnostic criteria. About 6-18% of adolescent girls are considered to have PCOS [2,3].

Because of the heterogeneity of the disease, there are ongoing investigations into new hormones, proteins, and cytokines that may help predict the diagnosis of PCOS [4,5]. These investigations can also clarify the pathogenesis and can find guiding biomarkers. Today, the biomarkers we used to confirm the diagnosis of PCOS are luteinizing hormone (LH), follicle-stimulating hormone (FSH), anti-Müllerian hormone (AMH), and androgens. AMH has some advantages over other hormones because its plasma level is stable during the cycles but the others should to be measured during the first 5 days of the cycle. AMH levels are also not influenced by the changes in the hypothalamus-pituitary axis. AMH is one of the predictors of ovarian reserve and shows a positive correlation with the preantral and small antral follicles [6]. Inhibin A (INHA) is another hormone studied in this regard and it is thought that it can be used for diagnosis in cases with PCOS [5]. Both INHA and inhibin-B (INHB) are synthesized from the granulosa cells of the ovaries. They are the bioactive forms of inhibins [7]. In the menstrual cycle, FSH peaks in the early follicular phase and this peak is thought to be the reason for the increased secretion of INHB from small antral follicles. While the increase in midcycle LH stimulates pre-follicular INHA secretion [8]. In light of this information, increment in INHB can be expected in patients with PCOS with many small antral follicles. As the follicle grows, INHA levels rise and are therefore likely to be normal or decreased in PCOS patients [9,10]. There are controversial results regarding INHB and INHA levels in PCOS [9,11].

In this study, we aimed to investigate the relationship between the clinical, anthropometric, laboratory, and ultrasound findings in adolescents followed up with the diagnosis of PCOS and to investigate the levels of specific biomarkers that may contribute to the development of PCOS.

Material and Methods

This retrospective cross-sectional study included 41 adolescent girls who presented to the Pediatric Endocrinology Clinic of Adana City Training and Research Hospital between January 2020 and December 2021 and were diagnosed with PCOS according to the clinical, laboratory and ultrasonographic findings [12]. Written informed consent was obtained from all participants and/or their families. The study was conducted in accordance with the Declaration of Helsinki. The ethics committee of Adana City Training and Research Hospital approved the study (Date: 2022-09-08, No: 2125).

Anthropometric measurements were obtained from patient records, and body hair growth was assessed using the Ferriman-Gallwey scale by a pediatric endocrinologist [13]. Standard deviation scores (SDSs) were calculated according to Turkish national pediatric data [14]. Body mass index (BMI) = weight (kg)/height (m^2) was calculated using the formula.

The free androgen index (FAI) is calculated based on levels of testosterone (T) and sex hormone binding globulin (SHBG) values [the formula FAI=100x(T/SHBG)]. Insulin resistance was calculated according to the hemostatic model (HOMA-IR) [(insulin [μU/mL] × glucose [mg/dL])/405] [15]. HOMA-IR > 2.5 was used to define insulin resistance (IR) [15]. Serum LH and FSH concentrations were measured using an immunochemical assay; dehydroepiandrosterone sulfate (DHEA-S) and T levels were measured using a radioimmunoassay (Ria) (Diagnostic Products Corporation, Los Angeles, CA, USA). 17-hydroxyprogesterone (17OH-P) and serum cortisol levels were measured with immunological Ria and Dia assays. SHBG levels were analyzed by IRMA, free testosterone levels were measured using a Ria, serum levels of INHA, INHB, and AMH (AL105-I) were determined by enzyme-linked immunosorbent assay (ELISA). Transabdominal US imaging of the pelvis was performed by a radiologist. If the ovary had 12 or more follicles, each 2-9 mm in diameter, it was defined as polycystic [16].

SPSS 25 (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, version 25.0. Armonk, NY: IBM Corp.) was used for statistical analysis. Results were reported as mean ± SD or median (min-max). Three different methods (skewness-kurtosis, variability coefficient and Kolmogorov–Smirnov values) were used for the normality tests of continuous variables, and it was considered that the findings in two of these three methods confirmed the normal distribution. Comparisons between groups were analyzed with parametric and non-parametric tests. The chi-square test was used to compare categorical variables, and Student's t-test was used to compare continuous variables. The Mann–Whitney U-test was used to analyze irregularly distributed parameters, and the Pearson and Spearman correlation parameters were used for correlation analysis.

Ethical Approval

Ethics Committee approval for the study was obtained.

Results

The mean age of the 41 adolescent girls included in the study was 15.62±1.43 years, and the mean age at first menarche was 11.98±1.32 years. According to World Health Organization obesity definition in children, a total of 68.2% of the patients were overweight (1≤ BMI SDS <2) or obese (BMI ≥ 2 SDS). Oligomenorrhea (90.2%) was the most common admission complaint, followed by hirsutism (63.4%), menorrhagia (7.3%), and polymenorrhea (4.9%).

When the patients with PCOS with and without hirsutism were compared, age at admission was higher and body mass index (BMI) was lower in the group with hirsutism, and the difference was significant (15.04 ± 1.61 years versus 15.95 ± 1.23 years; p=0.048 and 25.79±7.21 kg/m2 versus 31.44±6.70 kg/m2; p=0.019, respectively). In the hirsutism group, alanine aminotransferase (ALT) and Tg levels were significantly lower (17.12±8.91 vs 26.5±13.5 U/L; p=0.014 and 73.29±27.09 vs 120.75±52.78 mg/dl; p=0.012), the INH-B level was significantly lower (66±45 ng/dl vs 107±37 ng/dl; p=0.038).

Clinical, anthropometric, and laboratory findings of patients with and without hirsutism with PCOS are shown in Table 1. When the patients with PCOS with hirsutism and the non-hirsutism PCOS group were compared, the frequency of USG findings compatible with PCOS was similar (62.5% vs. 61.5%; p> 0.05). When the ultrasound findings were compared with the group compatible with PCOS and those incompatible with PCOS, a significant difference was found in terms of AMH level (11.0±7.3 mcg/L vs 5.9±2.8 mcg/L; p=0.032).

When overweight or obese (BMI SDS ≥ 2) patients with PCOS were compared with the normal-weight PCOS group, a significant difference was found in terms of high-density lipoprotein (HDL) and triglyceride (Tg) levels (42.94 ±8.95 mg/dl vs 55.22±14.92 mg/dl; p=0.011 and 111.31 ±46.16 mg/dl vs 58.77±12.24 mg/dl; p<0.001, respectively). When the overweight or obese PCOS group and normal-weight PCOS patients were compared, a significant difference was found in terms of HOMA-IR and insulin levels (5.02±6.22 vs 1.52±0.65; p=0.009 and 22.13±26.03 uU/mL vs 7.43±3.08 uU/mL; p=0.009) and age of first menstruation (11.50±1.39 vs 12.75±0.75 years; p=0.008, respectively). The clinical, anthropometric, and laboratory values of the overweight or obese patients and the patients with normal weight PCOS are shown in Table 2.

Compared with the PCOS group with high HOMA-IR levels (≥2.5), the PCOS group with normal HOMA-IR levels had significantly higher BMI values (32.47±6.92 kg/m^2 vs 23.22±4.58 kg/m^2; p<0.001), HDL was lower (41.64±8.64 mg/dl vs 56.08±13.18

Table 1. Clinical, anthropometric and laboratory findings in patients with PCOS with/without hirsutism

	PCOS group with hirsutism (n:26)	PCOS group without hirsutism (n:15)	p
Age (years)	15.95±1.23	15.04±1.61	0.048
First menstrual age (years)	11.97±1.18	12.00±1.61	0.96
BMI (kg/m^2)	25.79±7.21	31.44±6.70	0.019
LH (mIU /mL)	12.28±9.88	10.81±5.95	0.6
FSH (mIU/mL)	6.25±2.24	5.46±1.74	0.24
LH/FSH	1.91±1.38	1.89 ±0.85	0.96
T (ng/mL)	0.81±0.36	0.74±0.23	0.51
fT (pg/mL)	2.14±1.16	1.97±1.56	0.7
FAI	3.08±2.94	1.61±1.12	0.25
DHEA-S (µg/dL)	302.34±148.10	257.31±121.47	0.32
SHBG (ng/mL)	46.33 ±41.74	62.94 ±56.92	0.44
17-OHP (ng/mL)	0.90 ±0.39	0.84±0.40	0.64
AMH (ng/mL)	8.60±6.79	8.59±4.48	0.99
INHA (pg/mL)	6.10±3.23	5.47±2.48	0.6
INHB (pg/mL)	66.08±45.99	107.16±37.31	0.038
Insulin (µU/mL)	17.32±27.31	16.61±8.72	0.92
HOMA-IR	3.80±6.51	3.83±2.12	0.98
HDL (mg/dl)	50.35±14.73	43.75±8.48	0.17
LDL (mg/dl)	94.82±10.21	103.66±17.01	0.092
Triglyceride (mg/dl)	73.29±27.09	120.75±52.78	0.012
Total cholesterol (mg/dl)	155.16±9.41	169.16±21.52	0.17
TSH (mIU/L)	1.80±0.93	2.09±1.11	0.37
fT4 (ng/dl)	0.87±0.10	0.85±0.08	0.47
fT3 (ng/dl)	3.51±0.57	3.86±0.47	0.55
AST (U/L)	19.91±9.30	22.60±5.57	0.32
ALT (U/L)	17.12±8.91	26.50±13.52	0.014

*Hirsutism is defined as Ferriman-Gallwey score ≥8, †PCOS: Polycystic ovarian syndrome, BMI: body mass index, LH: luteinizing hormone, FSH: follicle-stimulating hormone, T: total testosterone, fT: free testosterone, FAI: free androgen index, DHEAS: dehydroepiandrosterone sulfate, SHBG: sex hormone binding globulin, 17OHP: 17-hydroxyprogesterone, AMH: anti-Müllerian hormone, INHA: inhibin-A, INHB: inhibin-B, AST: aspartate aminotransferase, ALT: alanine aminotransferase, HDL: high-density lipoprotein, LDL: low-density lipoprotein, TSH: thyroid stimulating hormone, fT4: free thyroxine, fT3: free triiodothyronine

Table 2. Laboratory findings in patients with PCOS with/without obesity

	Overweight or obese PCOS (n:26)	Normal weight PCOS (n:13)	p
First menstrual age (year)	11.50±1.39	12.75±0.75	0.008
LH (mIU/mL)	10.09±6.25	14.50±10.97	0.11
FSH (mIU/mL)	5.49±2.26	6.67±1.41	0.095
LH/FSH	1.72±0.87	2.14±1.44	0.27
T (ng/mL)	0.79±0.37	0.79±0.24	0.99
fT (pg/mL)	2.18±1.58	1.95±0.47	0.51
FAI	3.22±3.45	2.21±0.69	0.39
DHEA-S (µg/dL)	283.82±158.44	295.52±108.29	0.81
SHBG (ng/mL)	48.96±49.57	43.04±25.89	0.74
AMH (ng/mL)	7.75±3.86	10.97±8.23	0.18
INHA (pg/mL)	5.33±2.64	7.11±3.22	0.14
INHB (pg/mL)	84.46 ±45.67	76.74±54.23	0.73
Insulin (µU/mL)	22.13±26.03	7.43±3.08	0.009
HOMA-IR	5.02±6.22	1.52±0.65	0.009
HDL (mg/dl)	42.94±8.95	55.22±14.92	0.011
LDL (mg/dl)	102.21±14.50	91.33±10.31	0.055
Triglycerides (mg/dl)	111.31±46.16	58.77±12.24	<0.001
Total cholesterol (mg/dl)	162.27±18.29	161.00±0.01	0.94
AST (U/L)	20.40±5.95	22.23±11.55	0.52
ALT (U/L)	22.48±12.85	17.33±8.11	0.21

*PCOS: Polycystic ovarian syndrome, BMI: body mass index, LH: luteinizing hormone, FSH: follicle-stimulating hormone, T: total testosterone, fT: free testosterone, FAI: free androgen index, DHEAS: dehydroepiandrosterone sulfate, SHBG: sex hormone binding globulin, 17OHP: 17- hydroxyprogesterone, AMH: anti-Müllerian hormone, INHA: inhibin-A, INHB: inhibin-B, AST: aspartate aminotransferase, ALT: alanine aminotransferase, HDL: high-density lipoprotein, LDL: low-density lipoprotein

Table 3. Laboratory findings in patients with PCOS with/without insulin resistance

	HOMA-IR high (n: 20)	HOMA-IR normal (n:20)	p
BMI (kg/m^2)	32.47±6.92	23.22±4.58	<0.001
LH (mIU/mL)	10.22±5.47	12.43±10.40	0.4
FSH (mIU/mL)	5.41±1.77	6.52±2.32	0.097
LH/FSH	1.82±0.80	1.83±1.37	0.07
T (ng/mL)	0.80±0.40	0.78±0.22	0.89
fT (pg/mL)	2.33±1.71	1.81±0.62	0.22
FAI	3.59±3.42	1.77±1.00	0.1
DHEA-S (µg/dL)	287.27±158.70	284.70±124.72	0.95
SHBG (ng/mL)	36.00±39.73	65.48 ±48.75	0.12
AMH (ng/mL)	8.38±4.01	8.87±7.77	0.83
INHA (pg/mL)	6.00±2.27	5.69±3.65	0.79
INHB (pg/mL)	93.54±46.53	67.49±44.60	0.2
HDL (mg/dl)	41.64±8.64	56.08±13.18	0.001
LDL (mg/dl)	103.94±14.12	90.75±9.48	0.009
Triglyceride (mg/dl)	108.00±49.07	71.58±30.70	0.031
Total cholesterol (mg/dl)	164.77±19.47	154.33±5.85	0.39

*PCOS: Polycystic ovarian syndrome, BMI: body mass index, LH: luteinizing hormone, FSH: follicle stimulating hormone, T: total testosterone, fT: free testosterone, FAI: free androgen index, DHEAS: dehydroepiandrosterone sulfate, SHBG: sex hormone binding globulin, 17OHP: 17- hydroxyprogesterone, AMH: anti-Müllerian hormone, INHA: inhibin-A, INHB: inhibin-B, HDL: high-density lipoprotein, LDL: low-density lipoprotein

mg/dl; p: 0.001, respectively), low-density lipoprotein (LDL) was higher (103.94 ±14.12 mg/dl vs 90.75 ±9.48 mg/dl; p: 0.009), Tg was higher (108.00±49.07 mg/dl vs 71.58 ±30.7 mg/dl; p: 0.031), and free T4 level was lower (0.83±0.09 ng/dl vs 0.887 ng/dl, p:0.049). The clinical, anthropometric and laboratory findings of PCOS patients with high HOMA-IR and normal HOMA-IR are shown in Table 3.

Treatment with combined oral contraceptives (COC) was initiated in 60% of patients and treatment with metformin in 20% of patients. The mean HOMA-IR and BMI of the PCOS group treated with metformin were significantly higher than those of the group treated with COC ((9.30±9.97 versus 2.44±1.62; p=0.005 and 36.43 ±8.19 kg/m2 versus 25.53±5.55 kg/m2; p<0.001, respectively).

Discussion

PCOS is an endocrinologic disorder that presents difficulties in the diagnosis and management of adolescents. Unlike oligomenorrhea in adults, which is defined as menstruation less than 9 times a year or fewer than 40 days, the term is inapplicable to adolescents [17]. In girls, oligomenorrhea (less than 8 cycles per year) is defined as 4 cycles in the first year after menarche, 6 cycles in the second year, or later than 45 days after menstruation [18]. Although oligomenorrhea is the most common reason for a visit to the doctor, it may be overlooked in the first periods after menarche [5]. In our study, as in the literature, oligomenorrhea was the most common complaint.

Inhibins are released from the ovaries, they are produced during follicular development and the relation of INHA and INHB with PCOS is still under investigation. INHB is secreted from small antral cells during the follicular phase [10]. Some studies have shown that INHB levels increase in patients with PCOS, more than healthy controls [19]. Overall, the results generally show that the level of INHB is not significantly altered in PCOS patients [9,11]. The correlation between BMI and INHB was studied, and a negative correlation has been reported between them, but it is not clear whether this relation is due to polycyctic ovaries or not [20]. In our study, we could not find a difference in obese and non-obese groups according to INHB levels. Interestingly;,we show that INHB level was found to be significantly lower in PCOS patients with hirsutism compared to PCOS group without hirsutism (66±45 ng/dl vs. 107±37 ng/dl; p:0.038). We think that studies investigating the relationship between INHB levels and androgens are needed.

AMH is secreted by granulosa cells. It is a member of the transforming growth factor beta family. AMH levels increase throughout childhood until puberty, during late ages of reproductive period it starts to decrease [21]. AMH is associated with the number of ovarian follicles and is used as an indicator of ovarian reserve [22]. In adult patients, it is used as a marker for PCOS, and it also guides us in the treatment process. It can be used as a biomarker of PCOS in adolescents, but results of studies are conflicting. However, most studies have shown that AMH levels are increased in patients with PCOS, whether statistically significant or not [23]. In our patient groups, AMH level was found to be higher in patients with morphology compatible with PCOS in ultrasonography (US) findings than in the group without it. Yetim et al., suggest using the cut-off point of AMH as 6.1 ng/mL for the diagnosis of PCOS and the mean level AMH in our study was 8.60±5.86 ng/mL [7]. It can be accepted as a positive correlation indicator with the level of AMH and the number of preantral and small antral follicles, and we believe that it will also help us in diagnosing of PCOS in adolescents [8].

Lifestyle interventions (preferably in the form of diet, less sedentary behavior, physical activity, and behavioral strategies) should be recommended for all individuals with PCOS and obesity to achieve reductions in weight, central fatigue, and insulin resistance. As expected in our study, most of our patients were obese/overweight and had insulin resistance, and were advised on lifestyle recommendations. Pharmacologic treatment recommendations for adolescents with PCOS include the use of a combined oral contraceptive pill (COCP) and/or metformin in patients with a definite diagnosis for symptom control or in adolescents with PCOS who are considered “at risk” [24]. In the treatment recommendations in the guidelines, metformin treatment was initiated in the group with significantly higher BMI and HOMA-IR levels [3] because it was thought to be preferable in PCOS patients with obesity and/or impaired glucose tolerance. Treatment with combined oral contraceptives (COC) was initiated in 60% of our patients and treatment with metformin in 20% of patients.

Limitation

This study had several limitations, including its retrospective nature, limited sample size, and lack of standardization of ultrasound. Since there was a study targeting PCOS patients, there was no control group, and therefore, biomarkers could not be evaluated in terms of PCOS.

Conclusion

In conclusion, in this study, low inhibin-B was found to be more prominent in PCOS patients with hirsutism, while AMH was found to be significantly higher in the group whose ultrasound findings were compatible with PCOS since it directly reflects the size of the ovarian primary follicle pool. Prospective, randomized controlled trials involving more patients are required to use these markers in the diagnosis of PCOS.

Scientific Responsibility Statement

The authors declare that they are responsible for the article’s scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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